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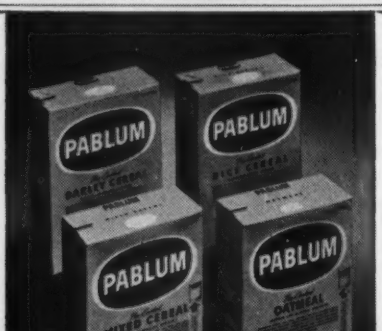

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A NEW CONCEPT FOR THE USE OF THE ELECTROPHRENIC RESPIRATOR (EPR)

John A. Doyle, M.D.
Joseph M. LoPresti, M.D.

The prime indication for the use of the tank-type respirator in poliomyelitis is in patients who have high spinal cord involvement with paralysis of the intercostal muscles and/or the diaphragm. In this group of patients, this type of respirator is live-saving. However, its use is attended by certain disadvantages which are directly related to a combination of two factors:

1. The confining space places definite limitations on nursing care, physiotherapy, and motion of the extremities.

2. Respirations are maintained primarily by negative pressure over the thorax and abdomen. Expansion of the lung parenchyma is limited, respiratory exchange is small, and hypoventilation occurs commonly.

The complications which may occur as a result of the first factor include: peripheral thrombosis and pulmonary emboli, nephrolithiasis, constipation and appendicitis, and decubitus ulcers; those which may accrue as a result of the second factor include atelectasis, pneumonias, fixation and fibrosis of the diaphragm, and acid-base imbalances. In addition, it is not unusual to encounter difficulty in inducing patients to rely on their own recovering ventilatory mechanisms.

Plum and Whedon[1] have recently utilized the rapid-rocking bed in an attempt to minimize these complications. Their results are encouraging; however, they met with some disturbing limitations. The most serious of these was the inability to utilize the rocking-bed in patients who were in the acute stage of the illness. In addition, the high cost of the rocking-bed might preclude its use in large-scale, severe epidemics. Thus, there is still a need for a device which would supply artificial respiration, minimize the complications of the tank-type respirator, and possess none of the limitations of the rapid-rocking bed.

Sarnoff has developed the electrophrenic respirator which by direct stimulation of the phrenic nerve produces active contractions of the diaphragm. The field of use of the EPR has not, as yet, been thoroughly investigated, but its scope is wide. To date, the electrophrenic respirator has been utilized with good results in poliomyelitis patients who have respiratory embarrassment on the basis of *central involvement.*[2] Theoretically, it would be an ideal adjunct in the management of patients who possess high spinal paralysis. There are numerous reasons for this concept:

1. The electrophrenic respirator is an available and relatively inexpensive instrument.

2. It can be utilized in the acute stage of the disease.

3. Tank-type respirator patients could be removed at regular intervals and subjected to electrophrenic stimulation for variable periods of time.

4. By producing active contractions of the diaphragm early in the disease, the danger of diaphragmatic fibrosis would be minimized.

5. Adequate lung expansion and ventilation could be achieved during the periods of electrophrenic stimulation. Theoretically, this would abort acid-base imbalances secondary to hypoventilation and prevent the respiratory complications which are due to lung parenchymal stasis.

6. During these periods, the nursing care and physiotherapy would be simplified.

7. Weaning of the patient from the tank-type respirator could be accomplished earlier, and the danger of psychological dependence would be lessened.

This year the number of cases and the severity of poliomyelitis in the Greater Washington area have been relatively low. For this reason, we have not had the opprotunity to fully evaluate our concept. It is presented so that other investigators in the field of poliomyelitis may carry the studies further.

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COMMON PEDIATRIC ORTHOPEDIC PROBLEMS

Special Report No. 218

Carl Berg, M.D.
Milton C. Cobey, M.D.
Allen Lloyd, M.D.
William J. Tobin, M.D.

INTRODUCTION

Allen Lloyd, M.D.

In the following discussions an attempt has been made to consider the common abnormalities encountered in the lower extremities of children. There are limitations to our knowledge. In some cases one cannot say whether or not treatment is necessary. When in doubt, it is safer to treat

the case, as long as the treatment involves no risk. There are more ways than one to skin a cat. Dr. Berg feels that the golden period has passed when the child reaches walking age with certain foot abnormalities. I am still pleased with results of education and treatment in the adolescent period. Certainly, in club feet and metatarsus varus, one cannot overemphasize the importance of complete over-correction and early and prolonged treatment. Success in these cases varies directly with the promptness of institution of treatment, degree of over-correction, and length of follow-up. The number of conditions considered by Drs. Cobey and Tobin and the thoroughness of the discussions were limited by available time, but both have covered their subjects in a remarkable manner.

In summary, it is felt by this discussor that one of the greatest contributions that the orthopedic surgeon can make is to advise in cases of doubt as well as to treat those conditions in children where deformity or malfunction of the musculo-skeletal system is obviously present.

It is the prime purpose of these papers to bring again to the attention of the pediatrician the importance of certain common orthopedic abnormalities which, in the normal course of events, may be overshadowed by more dramatic and dangerous conditions.

FOOT PROBLEMS

Carl Berg, M.D.

Except for specific diseases characterized by generalized joint rigidity, of which arthorgryposis multiplex congenita is the outstanding example, the tissues of the infant are pliable and may be moulded. Parts may be held in a favorable position for proper growth and development.

The extent to which alteration in development can be controlled is well illustrated in the deformities which are deliberately produced by primitive peoples. The bound foot of the Chinese woman is a grotesque perversion of structure. It scarcely resembles a human foot. Some aboriginal tribes bind the heads of infants until they assume the form of a cone. We employ the same principles of moulding and guided development to produce normal structures from initially deformed ones.

The two most common conditions for which I see children in the office are weak feet and rotational deformities of the lower extremities. Relaxed feet is only one of the manifestations of general ligamentous relaxation. The child of the generally relaxed type shows a hyperflexible spine and hyperextension of the elbows and knees. The muscles tend to hypotonia. Although the deficiencies in the weak foot are inherited, I believe that the future of this type of foot can be definitely altered by early and persistent treatment. Observation has confirmed this belief.

It is true that weak muscles or ligaments are further weakened by continued stretching. It is also true that if muscles or ligaments are main-

tained in a shortened position they will develop permanent shortening. Therefore, if the extremely relaxed foot of the infant dorsi-flexes and everts excessively, improvement can be accomplished by holding the foot in inversion and plantar flexion in plaster casts. When the desired change has been accomplished, retention may be maintained by the use of posterior plaster splints at night. The infant with weak feet will dorsi-flex in eversion when the foot is stimulated. The invertors are not exercised and become relatively weaker than the evertors. Exercise to strengthen the inverting muscles is required. By controlling the direction of muscular movements we may force the infant to perform proper exercise. Normal kicking movements take care of this. It is only before the age of walking that we can take advantage of this opportunity. After the child has begun to walk the automatic movements cease and with them practically all hope of success with any exercise program.

I should like to stray for a moment from the specific subject at hand to say a word about exercises as part of the rehabilitation program. By and large, any benefits from exercises which the patient is supposed to do may be discounted heavily. This is true of the adult and child. Most of our programs are two-part affairs, one which we do and the other which the patient is supposed to do. The first part is usually conscientiously done, the second is almost invariably neglected. Intentions are good but the spirit is weak. Hence the importance of guided automatic exercise in the early months of the child's life.

Exercises are controlled by holding the extremities in internal rotation and inversion by attaching the feet to a board or metal bar. When the infant kicks the feet are moved in the right direction. After the age of walking, the feet must be properly supported by shoes so that weak ligaments will not be under constant stretch.

Rotational deformities of lower extremities are very common, internal rotation much more than external. Mild cases may spontaneously improve, but severe ones certainly will not. Persistent internal rotation constitutes a real deformity in adult life, and is especially disfiguring in a woman. External rotation tends to cause pronation of the feet in addition to producing an unsightly gait. These deformities develop because of persistent maintenance of the faulty position.

The majority of infants spend most of their time in the prone position. If, at birth, there is a tendency to internal rotation this propensity is increased and perpetuated by constant retention of this position. This leads to shortening of the internal rotators of the hips making normal external rotation at this level impossible. Usually, in addition, there is internal torsion of the tibias. On occasion, there is adduction of the forefoot. If the child is walking, it may be readily noted at what levels the deforming

influences are present. The patellas point inward indicating that the hips must be involved. The feet are rotated more than the knees indicating internal torsion of the tibias. Before the age of walking, it is noted that external rotation of the hips is limited. When the knees are pointed straight ahead, the feet point together. Everything that these children do is done with the extremities in the mal position. They lie that way, they crawl that way, and they walk that way. The remedy seems obvious. Applying a night splint holding the extremities in external rotation will overcome the defect. It has long been common practice to apply a wedge to the outer side of the shoes in the walking child to overcome internal rotation. This device is not effective since the deforming forces are not in the foot. Another device often tried but very seldom effective is the so-called "twister strap" which consists of a strip of adhesive tape wound spirally around the leg twisting it into external rotation. This is too difficult for the child to wear and hampers his normal activities. He will usually develop a method of walking which defeats the purpose of the strap. The deformity is maintained by the lying position so that it is much more logical to apply night splints. Whenever night splints will accomplish correction they are preferable to any device worn during the daytime.

There is another deformity which strangely is appearing to be on the increase. This is the so-called "metatarsus-varus primus" or "forefoot adduction." It is sometimes referred to as one-third of a club foot since the true club foot possesses forefoot adduction as one of its deformities. Increasing awareness of the deformity is probably one of the reasons why we see more of it but this does not account entirely for the increased incidence. Dr. Hiram Kite, who is the pioneer in the treatment of club feet, feels certain that the incidence of metatarus-varus is increasing, while that of true club foot is diminishing. This is a definite boon since the treatment and end results of metatarsus-varus are much more satisfactory.

In this deformity, all the elements of the foot are in normal relationship except the metatarsals which are adducted or swung-in with respect to the tarsal elements. The foot shows a distinct concavity on the medial side with a tendency of the big toe to adduct excessively. Failure to correct this deformity early, results in severe deformities of the foot in later life. When regular shoes are worn, the first toe is forced into the valgus or abducted position. This leaves the first metatarsal head unduly prominent on the medial side of the foot. Excessive pressure on the head of the metatarsal gradually produces an exostosis which finally results in the formation of a typical bunion with hallux valgus. It is altogether likely that the incidence of bunions will diminish in years to come because the basic deformity is being corrected in infancy.

The deformity should be corrected as soon as it is noted. Correction is

accomplished by moulding in plaster casts. Following plaster correction, out-flare shoes should be worn to maintain normal position. In milder cases the special shoes may be omitted. Manipulation of the feet on the part of the parents, if conscientiously done, will prevent recurrence in the less resistant cases. In those cases in which they are required the special shoes should be worn at night until all tendency to recurrence has disappeared.

Let me say just a word in regard to the wearing of night splints during infancy and early childhood. Parents naturally rebel, feeling that the restraint will be a frustrating experience to the child. This is not at all true. It is very rare to find a child who does not accept splinting without complaint. Experience in hundreds of cases has furnished adequate proof of this.

More errors are made by failure to observe than because of ignorance of those conditions which should be observed. External vigilance is the price of success.

GROWTH DISTURBANCES ABOUT THE KNEE

Milton C. Cobey, M.D.

The distal femoral epiphysis is the largest in the body. Like the upper tibial epiphysis, it is present by x-ray at birth, and is usually active and not fused until the nineteenth year. The ossification center of the patella, on the other hand, is not visible by x-ray until four years of age and has attained adult size at fourteen years of age.

The greatest longitudinal growth of any bone is carried out by the distal femoral epiphysis and, second to it, by the proximal tibial epiphysis. These epiphyses are the largest and produce the most growth. There is little wonder then, that they have growth distrubances.

The most common abnormality is in the tongue of the upper tibial epiphysis which may become fragmented and give trouble all through life unless treated early and well. This is often called Osgood-Schlatter's disease or osteochondritis of the tibial tubercle. When it first becomes symptomatic, good care will correct the disease and prevent further trouble. This must include absolute rest. Nothing has been found so effective as the plaster cast. The high protein, low free sugar, high vitamin, and green vegetable diet, as recommended by Dr. Risser, will be most beneficial with the cast.

Infantile tibial torsion is very common. This is a twist of the tibia inward or outward. The exact cause is obscure, but several reasons are advanced. Among these are heredity; uterine position; allowing the infant to sleep too much on either the back or abdomen; or in the older child, static uncorrected foot deformities. Splints, braces, or shoes will correct

the younger and milder cases, while high tibial osteoclasis or osteotomy is required for the older and more deformed cases.

Bowing deformities of the tibia are possible in all directions. Posterior bowing of the tibia, when allowed to persist, will produce a genu recurvatum in the adult bone that can be troublesome. From the number of adults who have a "back-knee" deformity, I believe that this is the most overlooked bowing deformity in infants. This is again correctible, but the older the patient, the more technically difficult the surgery and the longer the period of disability. In the infant and young child, the use of casts and braces often will correct this.

Another deformity is the anterior bow of the tibia or "sabre-shin" (nonluetic). This is often associated with the more severe angulation that occurs in utero or just after birth. It occurs at the junction of the distal and central ossification centers; is either a congenital nonunion or congenital angulation, and is present at times when there is a congenital absence of fibula. This deformity of anterior bowing is more easily recognized and is usually corrected early by casts and braces or osteoclasis.

Medial or lateral bowing of the femoral and/or tibia is called "bowlegs and knock knees." Genu varum or genu valgum are the medical terms.

The causes of all types of bowing deformities are again inherited tendencies. Parents will pass along this deformity often associated with pes planus or pes cavus congenital abnormality. The patient with dyschondroplasia tendencies leading toward dwarfism also develops bowing. Rickets, of course, accounts for a number of these cases. The child with mild rickets may have a pot belly, right knock knee and left bowleg. Rickets does exist today. Not so many children need osteotomy or osteoclasis as in past years for rachitic bowing, but many mild cases still appear. It is a known fact that no one vitamin D preparation is good for all children. Some will not absorb the oil soluble vitamin and must have the water soluble preparation.

For example, in a family of four boys, ages 7, 5, 4, and 3, all children were under good pediatric care and all took oil soluble vitamin D. Ages 7 and 4 developed no bowing. Ages 5 and 3 developed such severe bowing of both femoral and tibiae that surgical correction of all four bones in each case had to be done. They were too old for osteoclasis. It is not possible to maintain correction if the femora and tibiae are osteotomized at the same operation. It was elected in these cases to osteotomize the tibiae and staple the femoral epiphyses. The correction of the tibia is obvious. The temporary cessation of growth of the lateral side of the femoral epiphyses by the staples after 6 months for the younger child and after 1 year for the older child demonstrates the correction obtained at the time the staples were removed.

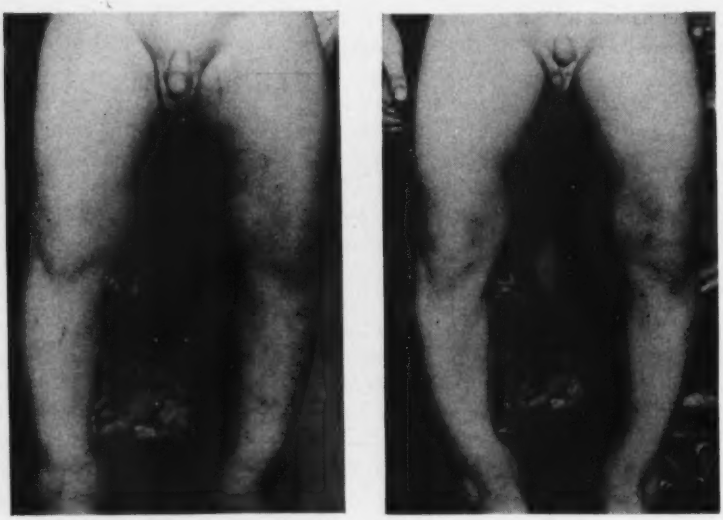

FIG. 1

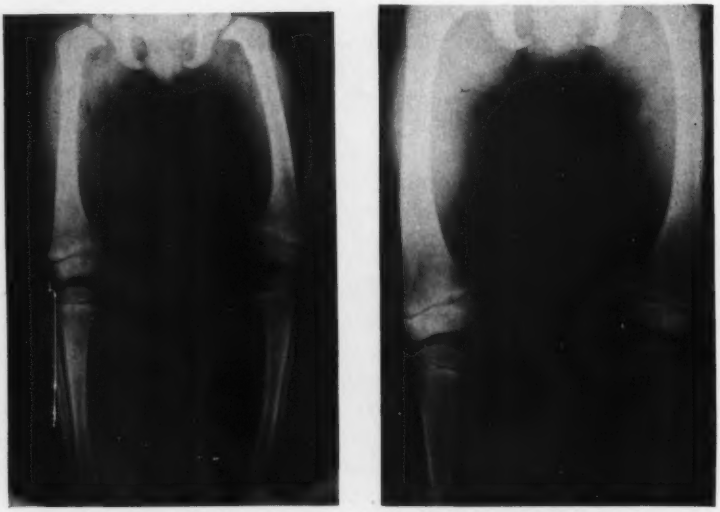

FIG. 2

One cannot explain this growth disturbance in these children. Could it be rickets or dyschondroplasia? A tendency toward dyschondroplasia could

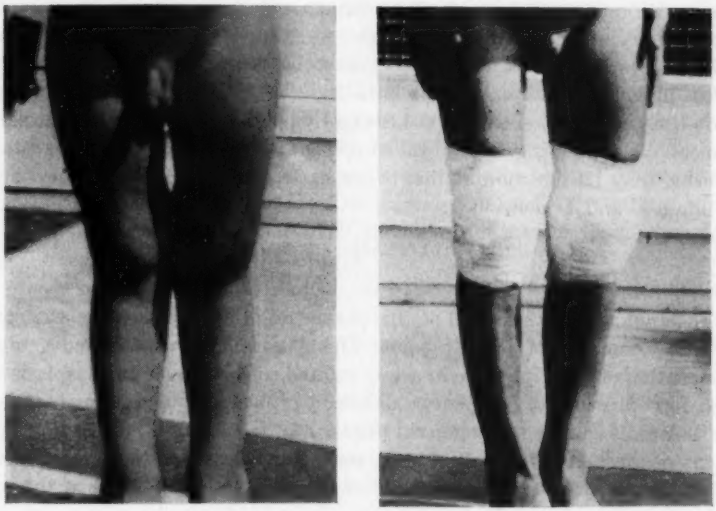

FIG. 3

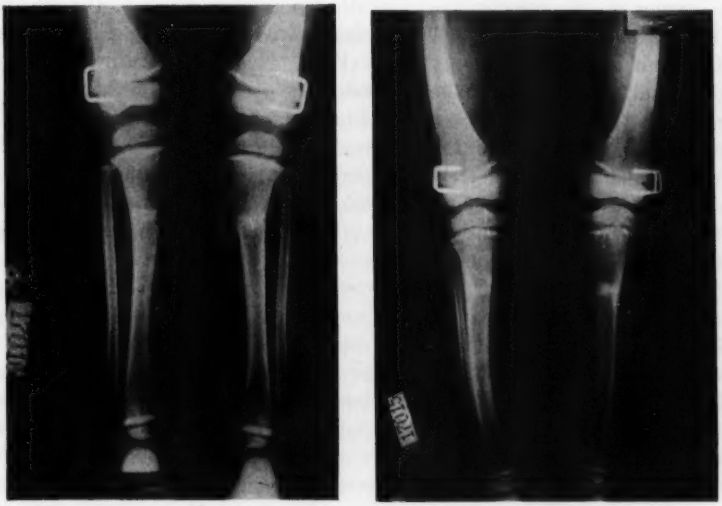

FIG. 4

play a part here. We use what vitamins we feel are best for the child. Modern civilization as it affects the rearing of children also helps to produce

these deformities. Babies get very fat on our present formulas. Grandmothers encourage them to stand and walk early. Parents are taught by every advertisement that children must be taught to walk, which is a gross untruth. Children walk as birds fly—when they are the correct age. Too-early stimulation to weight bearing by playpens, walkers, and strollers causes a lot of bowing. The so-called epiphysitis of infancy producing tibial bowing may be the combination of too-early weight bearing, obesity, and vitamin C and D deficiency.

CONGENITAL DYSPLASIA OF THE HIP JOINT

William J. Tobin, M.D.

It is not within the scope of this presentation to discuss all of the congenital anomalies of the hip joint. The classical, well defined congenital dislocation of the hip, while in a way related to the congenital dysplasia of the hip, presents a completely different problem. The significant basic pathological change in congenital displasia of the hip is a lack of development of the roof of the acetabulum and a retardation of the development of the femoral head. Oftentimes, there is a classical dislocation of the opposite hip. In true congenital displasia of the hip joint, there is no definite dislocation of the hip. Credit for emphasis on the early recognition and treatment of congenital displasia of the hip must be given to the late Dr. Vernon Hart.[1, 2] Dr. Hart felt that the defect had a genetic or biochemical etiology or a combination of the two. Putti[3, 4] brought out evidence that congenital subluxation of the hip exists as a distinct clinical and x-ray entity. Putti also emphasized the early recognition and treatment of this condition in infancy before weight bearing begins. Howorth and Smith[5] mention that among their cases, 7 per cent were only subluxated and 93 per cent could be classified as complete congenital dislocations. McCarroll and Crego[6] discussed this condition as one of the three types of dislocation of the hip.

It is the purpose of this discussion to emphasize the early clinical findings and roentgenographic changes that are present in congenital dysplasia of the hip. The condition should be recognized before the child begins to bear weight. After the infant begins to bear weight and walk, limping, shortening of the extremity, and telescoping can easily be recognized. It is before this time that the diagnosis should be made and treatment begun.

One of the earliest clinical objective findings in these cases is a limitation of abduction in the involved hip. This is due to the tightness of the adductor muscles and tendons. The pediatrician should, in his routine examination of the newborn infant, test the degree of abduction of the hips in both extension and in flexion. It is felt that pediatricians have not sufficiently

emphasized this aspect of the examination of the newborn infant. This part of the examination requires very little additional time and may enable the pediatrician to recognize a case of congenital displasia of the hip which otherwise will be missed until the patient begins to stand or walk. The presence of an additional fold in the gluteal region is suggestive, but not diagnostic, of congenital dysplasia or dislocation of the hip. It is felt that when an extra fold does exist, the condition should be further investigated by roentgenographic studies. The roentgenographic shadow of the head of the femur is not present at birth and does not appear definitely until the third or fourth month. Oftentimes, the displasia or lack of development of the acetabular roof can be determined by x-rays at an early date. If not conclusive, suspicion of some change in the angle of the acetabular roof certainly warrants additional x-rays at monthly or bi-monthly intervals.

It is felt by some that weight bearing causes the partially subluxated hip to progress to complete dislocation. Others feel that the dysplastic hip or partially subluxated hip remains as such throughout life. Hart mentioned that the possibilities in such a case could be defined as follows: 1. a normal head would progress with no difficulty whatsoever; 2. one may see a flat socket with no displacement; 3. a persistent subluxation may exist; 4. a complete dislocation may later present itself; 5. there is some malformation of the femoral head resulting in the flat head of a coxa plana type of head.

As mentioned in the roentgenographic picture of these cases, there is a shallow acetabulum, a retardation of the ossification of the femoral head, and a delayed closure of the ischiopubic junction. Also, some upper displacement of the head is seen. There is some distrubance of Shenton's line, a line forming an arc between the inferior neck of the femur and the inferior aspect of the pubic bone. In congenital dysplasia of the hip with some upward displacement of the head, there is a definite disturbance in the continuity of this arc. Figure 5 shows a congenital dysplasia of the right hip.

Treatment in Congenital Dysplasia of the Hip: Treatment should be begun as soon as the diagnosis is made. Because of the tightness of the adductor tendons, the involved hip should be kept in a position of abduction in order to stretch the tight adductor tendons and also to lever the head into the acetabulum. It is the presence of the head in the acetabulum that stimulates ossification of the nucelus of the femoral head and a redevelopment of the acetabular roof. The abduction splint of Frejka enables the hips to be held in a position of abduction, yet permits the patient to move his hips and even to walk in the splint. The Frejka splint (See Figure 6) consists primarily of a wide pad that fits in the groin and is attached over

FIG. 5. Female age six and one-half months with congenital dysplasia of the right hip. Note underdevelopment of the femoral head, dysplasia of the acetabulum, widening of the hip joint and disturbance of Shenton's line (continuation of arc formed by inferior aspect of the superior ramus of the pubic bone). Also note slight upward displacement of the femoral shaft and widening of the epiphysis of the right pubic bones.

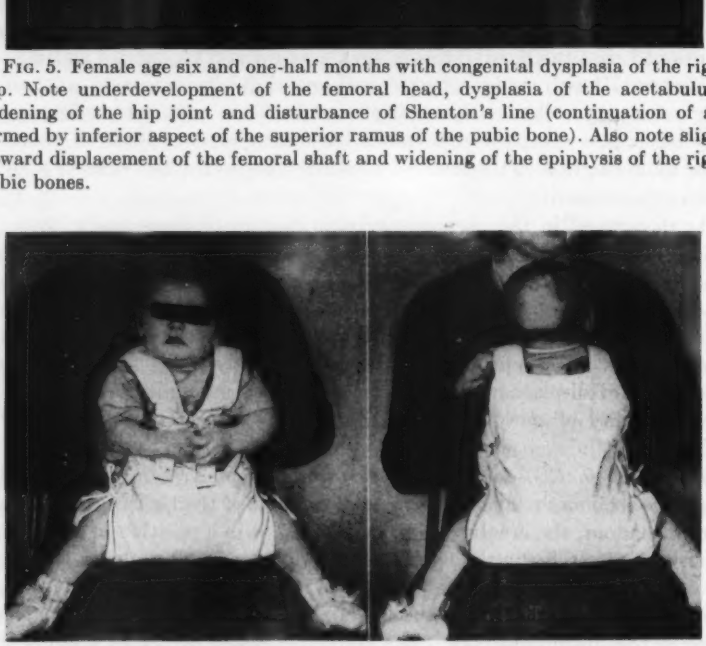

FIG. 6. Frejka Splint, front and back views. This consists of a wide pad, covered with a water-proofing material, keeping the hips in wide abduction. It attaches over the patient's shoulders and is adjustable. As the adduction contracture is overcome, increased abduction can be attained.

the infant's shoulders to keep it in place. The splint permits the hip to be kept in a position of abduction, and, as mentioned above, it is the presence of the head in the acetabulum that seems to stimulate growth of the head of the femur. This splint should be worn until there is roentgenographic evidence of redevelopment of the acetabular roof and the head of the femur. Roentgenograms should be taken at monthly or bi-monthly intervals until this redevelopment has been definitely established. This may require from four to six month or longer wearing of the Frejka splint before sufficient redevelopment of the acetabular roof and head can definitely be established.

In summary, an attempt has been made to emphasize the importance of early recognition and treatment of congenital dysplasia of the hip. Time does not permit a detailed discussion of the complete dislocation of the hip.

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SUGGESTED MANAGEMENT OF PYOGENIC MENINGITIS

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The following management routine for pyogenic meningitis has recently been adopted at Children's Hospital. Admittedly, the choice of optimal drug therapy still remains in a state of flux especially in pneumococcic and H. influenzae. However, the following regime was worked out primarily because of the confusion resulting from the plethora of antibiotics and the difficulty in deciding which drug or combination of drugs would offer a patient with pyogenic meningitis the benefit of maximum therapy. Since the inception of this regime, it has been of considerable help to the house staff in standardizing therapy in a complex disease.

DIAGNOSTIC PROCEDURES IN SUSPECTED CASES OF PYOGENIC MENINGITIS

I. Lumbar Puncture:
Smear, culture, cell count, chemistries (protein and quantitative sugar).
II. Blood culture.
III. Nasopharyngeal culture.
IV. Ear culture (if aural discharge present or myringotomy is done).
V. Petechial smear (always perform when petechiae are observed).
VI. Complete hemogram.
VII. Type and cross match for present or future needs.

TREATMENT OF CHOICE

I. Meningococcus Meningitis
(Combination sulfadiazine and chloromycetin recommended).
A. Sulfadiazine
1. Clysis Na sulfadiazine (1% solution) in 5% glocose with Hartmann's solution—0.25 to 0.5 gm./kg/24 hrs. One-half total dose stat, then 1/3 daily dose Q8° thereafter.
2. Change to oral sulfadiazine as soon as feasible.
B. Chloromycetin (intramuscular and oral).
1. *Intramuscular chloromycetin:* add 4.6 cc. saline to 2 gm. ampoule of intramuscular chloromycetin. Shake vigorously. Give 100–150 mgm./kgm. body weight every 12 hours during acute phase and every 24 hours during convalescent phase.
2. *Oral Chloromycetin:* Should be used after acute phase has subsided.
II. Fulminating Meningococcemia with Meningitis (Waterhouse-Friderichsen Syndrome).
A. Sulfadiazine, penicillin and chloromycetin—to be employed in combination.
B. Cortisone (See under Supportive Therapy).
III. H. Influenzae meningitis.
A. Combination Chloromycetin and sulfadiazine recommended.
1. Chloromycetin, intramuscular and oral. (same as in Meningicoccus Meningitis).
2. Sodium Sulfadiazine by clysis during acute phase. Change to oral sulfadiazine during convalescent phase. Dosage 0.25 to 0.5 gms./kg/24 hrs.
B. Streptomycin to be used only when failure to respond to above regime.

C. H. Influenzae antiserum:
To be employed in conjunction with sulfa and chloromycetin only in critically ill patients. It should be necessary only in rare cases.

D. No intrathecal administration of drugs recommended.

IV. Pneumococcal Meningitis

A. Combination penicillin and sulfadiazine recommended.

1. Sulfadiazine:
Same as in H. Influenzae meningitis.

2. Intramuscular Penicillin.

a. Dose 500,000–1,000,000 units aqueous penicillin stat intramuscularly; then 500,000 q2° during first 24°; then 500,000 q3° thereafter during acute phase.

b. Procaine penicillin (SR - Parke Davis) 400,000 q8° after acute phase has subsided.

c. Penicillin not to be administered intrathecally unless patient is in extremis. In latter instance, use 5000 units intrathecally per day in addition to previously noted treatment.

V. Staphylococcal Meningitis
Same treatment as Pneumococcal Meningitis.

VI. Streptococcal Meningitis
Same treatment as Pneumococcal Meningitis.

VII. E. Coli Meningitis
Same treatment as H. Influenzae Meningitis.

VIII. Meningitis of Doubtful Etiology.
If causative organism is doubtful during first 24 to 48 hours (i.e. in instances when smear is negative and cultural results awaited), a combination of parenteral sulfa and intramuscular chloromycetin is recommended initially.

A. If organism is then identified as *meningococcus*, continue sulfa drug and chloromycetin.

B. If organism is then identified as *H. Influenzae*, continue sulfa and intramuscular chloromycetin.

C. If organism is then identified as *pneumococcus*, start penicillin; continue sulfa drug, and omit chloromycetin.

IX. Mixed Meningitis

A. H. Influenzae and Meningococcus:
Combination sulfa and chloromycetin.

B. H. Influenzae and Pneumococcus:
Combination sulfa, penicillin, and chloromycetin.

C. Pneumococcus and Meningococcus:
Combination sulfa, penicillin, and chloromycetin.

SUPPORTIVE THERAPY

I. Parenteral fluids. Initial hydration is important.
II. Blood transfusions.
III. Oxygen.
IV. Cortisone—200 mgm. stat by injection and adrenal cortical extract 5 cc. or more q2h if patient in extremis with Waterhouse-Friderischsen syndrome.

FOLLOW-UP LABORATORY WORK

I. Spinal Punctures.
   A. Meningococcus:
      Repeat spinal tap prior to discharge from hospital.
   B. Pneumococcus and H. Influenzae.:
      Repeat spinal tap within 48 hours after treatment has been started and again prior to discharge. May be repeated more frequently if necessary for orientation.
II. Sulfa Levels.
   A. Daily during first three days; thereafter, every second day until sulfa is discontinued. Obtain just before next dose of drug is given.
   B. Optimal sulfa levels.
      1. Meningococcal—6–10 mgms.%.
      2. H. Influenzae—15–20 mgms.%.
III. Chloromycetin Levels.
   A. Daily determination (5 cc. clotted blood) just before next dose is given.
   B. Optimal chloromycetin level—15 to 30 mcgms./cc.
IV. Sensitivity of Organism.
   A. Routine chloromycetin sensitivity of pneumococcus, H. Influenzae and E. coli organisms.
   B. Routine penicillin sensitivity of pneumococcus, staphylococcus and streptococcus organisms.
V. Urinalysis
   A. Daily during acute phase of disease.
   B. Observe for oliguria and hematuria.
VI. Complete blood count.
   A. Every second day during therapy.
   B. Observe for leukopenia when chloromycetin and sulfa are employed.
VII. Eosinophile Count
   To be done q4h in case of suspected Waterhouse-Friderichsen syndrome or as often as indicated.

VIII. Subdural puncture
If poor response within 72 hours after treatment is started.
IX. Head circumference measurement—daily.
X. Neurological consultation—prior to discharge with arrangement for follow-up.

DURATION OF THERAPY IN PYOGENIC MENINGITIS

Continue for seven to ten days after temperature has returned to normal and spinal fluid rendered sterile.